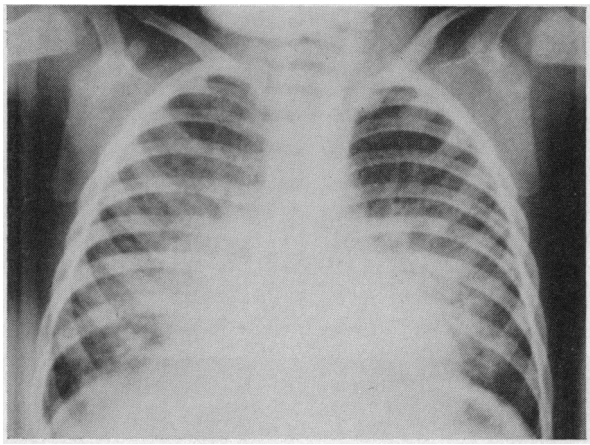

Figure 3.—Film taken on tenth day of illness showed extension of pneumonic process, although clinical condition was much improved.

the patient appeared moribund. He was unresponsive and febrile, and the respiratory rate was still 80 to 100. At this time administration of prednisolone, 20 mg intramuscularly every six hours, was begun.

Within 24 hours there was noticeable improvement in the patient's condition. The temperature decreased, becoming normal on the tenth day. Respirations slowed to 50 a minute, be became active and responsive and began taking fluids by mouth quite well—this although a chest film taken the tenth day showed extension of the pneumonic process (Figure 3). Clinical improvement continued until the nineteenth hospital day, when fever and leukocytosis recurred and rales were heard at the left lung base. Although an x-ray film of the chest showed some clearing, it was believed that the recurrence of symptoms was owing to bacterial pulmonary infection, and administration of chloramphenicol, which had been discontinued, was resumed (chloramphenicol palmitate, 60 mg by mouth every 6 hours). Within 24 hours the patient was again afebrile and his condition improving once more.

On the twenty-seventh day, severe diarrhea developed suddenly, then quickly subsided without specific therapy. Stool cultures showed only normal flora. The patient was finally discharged on the thirty-second hospital day with prescription of iron by mouth (Fer-in-sol, 0.3 cc twice daily). He was in excellent condition although x-ray films still showed some perihilar infiltration. The patient did well thereafter and a subsequent roentgenogram showed the chest completely cleared.

DISCUSSION

The accidental poisoning of children by the ingestion of petroleum distillates is still a distressingly frequent occurrence in this country. Among these compounds the one known as mineral seal oil produces a high incidence of pulmonary complications, as described by Griffin and co-workers.[1] Old English Furniture Polish contains over 90 per cent mineral seal oil.

In reviewing the literature we were able to find little information on the use of steroids in this condition. The Cooperative Kerosene Poisoning Study[3] dealt primarily with the technique of gastric lavage, and with the question of whether it should be used at all. Steroids were mentioned only in passing, with no recommendations about their use. In the case reported by Mayock and co-workers,[2] in which adrenal steroids were used, the patient was an adult with chronic pulmonary changes and the problems were unlike those in the case herein reported. However, Mayock cited reports by Nassau in the German literature and suggested that further evaluation of steroids in acute kerosene poisoning would be worth while.

SUMMARY

A child severely ill with chemical pneumonitis was treated with adrenal steroids in addition to meticulous supportive care, and dramatic improvement followed.

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Accidental Cutaneous Coccidioidal Infection in an Immune Person

A Case of an Exogenous Reinfection

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RARELY DOES COCCIDIOIDOMYCOSIS begin with the skin as a primary site of infection. Most cutaneous lesions result from the dissemination of organisms from some earlier, perhaps unrecognized, primary pulmonary infection. In a review of the literature, we found five clear-cut cases of accidental cutaneous coccidioidal infection, occurring in persons not previously immune, through an earlier primary in-

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fection. Wilson[7,10] referred to four additional unreported cases in man as well as three cases in dogs that infected themselves through injuries in their paws while digging in animal burrows contaminated with Coccidioides arthrospores. Only a single footnote reference could be found reporting primary cutaneous infection in a previously immune subject.[5]

In this report we are presenting a second case of an accidental cutaneous coccidioidal infection in a person previously immune as demonstrated by an earlier positive reaction to skin test.

REPORT OF A CASE

On September 21, 1962, a Caucasian man, a 12-year resident of the San Joaquin Valley who six weeks previously had had a coccidioidin skin test that was 2+ at 1:100, was inoculating a number of mice with a suspension of *Coccidioides immitis* arthrospores which produced 4×10^3 colonies per ml on mycosel agar. One of the mice, pulling free of the operator's grasp, caused him to flinch enough to jar the hypodermic needle forward, barely puncturing the skin at the side of the middle phalanx of his left index finger. An effort was made to induce bleeding from the puncture but only a very tiny drop of blood could be expressed. The puncture area was then rubbed vigorously with formalin. This was the only treatment at the time. Four days later a tiny induration could be felt over the slightly tender site of inoculation. During the following few days a small firm nodule with swelling developed but tenderness did not increase. By October 7 (17th day) a small white-centered pustule about 2 mm in diameter had formed. Leukocytes at this time numbered 8,700 per cu mm of blood, with 54 per cent neutrophils, 46 per cent lymphocytes but no eosinophils. The red-cell sedimentation rate was 11 mm in one hour. Material aspirated from the pustule was examined microscopically and cultured on blood agar plates. The microscopic preparation showed a very few mature spherules. Two had ruptured and the endospores appeared to be crenated or shriveled. One colony of fungus grew from the blood agar plate. This was transferred to a fresh mycosel plate where it grew colonies typical of "*C. immitis*." A mouse inoculated from this growth died in ten days with typical pathologic changes and spherule production.

On October 8 local irritating pruritis had developed around the site but there was no lymphadenopathy or lymphangitis. Two days later urticaria developed in the palms of both hands and then spread to all parts of the body. Antihistamine therapy did not alter the persistent urticaria, which lasted for six months. On the 32nd day the lesion,

TABLE 2.—*Cases of Accidental Cutaneous Coccidioidomycosis Recorded in the Literature*

Author and Year Reported	Sex Age Race	Site of Infection	Coccidioidin Skin Test Prior to Infection	Clinical Features	Precipitin and Complement Fixation	Specific Treatment Given	Outcome
Guy, Jacob,[1,2] 1926, 1927	Male, 36 yr.*	Pricked rt. thumb with cactus thorn	Test not available then	Ulcer with regional lymphangitis and lymphadenitis	Test not available then	None	Disseminated in six months
Wilson, Smith, Plunkett,[3] 1953	Male, 32 yr., White	Proximal phalanx rt. middle finger	Non-reactive	Same, plus fever and malaise	Pptin. 4+ in 1:10. C.F. 4+ in 1:2	None, except excision of lesion	Recovered except for persistent nodes,
Trimble, Doucette,[6] 1956	Female, 29 yr., White	Middle phalanx lt. 3rd finger	Non-reactive	Ulcer with lymphangitis and lymphadenitis	Pptin. 4+ in 1:40. C.F. 2+ in 1.2	None except excision of lesion	Recovered except for persistent nodes
Wright, Newcomer, Nelson, Wilson,[7,8,10] 1959, 1960, 1963	Male, 29 yr., Japanese	Lt. index finger	Non-reactive	Same, plus fever and malaise	Pptin. 4+ in 1:10. C.F. 2+ in 1:2	None, except excision of lesion	Recovered except for persistent nodes
Harrell, Honeycutt,[3] 1963	Male, 18 yr., White	Punctured lt. thigh with cactus thorn	Non-reactive	Ulcer at punctured site and lymphadenopathy	C.F. Negative (done several months after injury)	None	Recovered except for persistent nodes
Tiggert (cited by Smith et al.);[5] 1961	.....*	Deeply into a wrist bone	Reactive	Local lesion only	No record	None, except excision	Recovered after excision
Patient reported in this paper	Male, 49 yr., White	Lt. index finger	Reactive	Local lesion and urticaria	Pptin. ± undiluted. C.F. 4+ in 1:16	None. Excision done 32nd day for examination	Recovered

*Indicates respective data were not recorded.

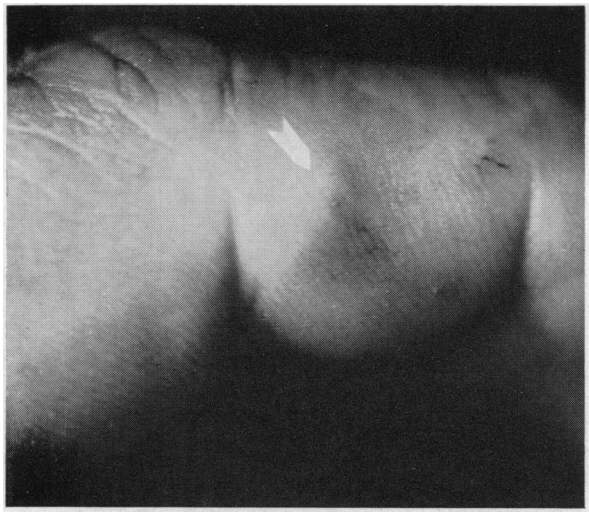

Figure 1.—Photograph of the lesion on finger (arrow) 32 days after inoculation.

a small granuloma, was photographed (Figure 1), and a biopsy specimen was taken from it. Half of the specimen was sectioned for histological examination and the remainder was ground and cultured on mycosel agar. The culture grew 33 colonies of "*C. immitis.*" The histologic report indicated an area in the corium in which there were strands of granulation tissue radiating between the bundles of collagen. Round cell infiltration was moderate and some areas had a few multi-nucleated giant cells.

Serologic tests for coccidioidal infection were carried out, beginning the fifth day after inoculation and at weekly intervals during the course of the infection (Table 1). The precipitin test was consistently negative. The complement fixation titer rose to 3+ at 1:16 dilution on the 25th day of infection and then fell to 3+ at 1:4 dilution on the 45th day. An x-ray film of the chest taken just before the accident was normal and there was no change in one taken at the end of the period of observation when the patient was well. Except for the generalized and prolonged urticaria and mild local lesion, there were no clinical manifestations. Because of the apparent dermal hypersensitivity manifested by the urticaria, no further skin tests were done.

TABLE 1.—*Complement Fixation Reactions In Present Case*

Weeks After Infection	Tube Dilution Results				
	1:2	1:4	1:8	1:16	1:32
1	2+	....	....	....	....
2	1+	....	....	....	....
3	4+	4+	....	....	....
4	4+	4+	4+	3+	....
5	4+	4+	4+	....	....
6	4+	4+	....	....	....
7	4+	3+	....	....	....

DISCUSSION

Of the six previously reported cases found in the literature, the first one was by Guy and Jacob.[1,2] Infection was through a cactus thorn prick in the finger. The second case was presented by Wilson and coworkers.[8] The skin as the portal of entry was well established. An interesting and complete report spanned the illness through recovery and four and a half years afterward. The authors presented criteria to be used in establishing that infection was by the cutaneous route. Trimble and Doucette[6] reported the third case, that of an accidentally infected microbiologist, and they compared features of the case with the criteria established by Wilson. In the fourth case, reported by Wright and coworkers,[9,10] a laboratory worker accidentally pricked his finger with an injection needle containing the tissue phase of "*C. immitis.*" Smith and coworkers[5] made mention, in a footnote, of a fifth case in which Tiggert observed a laboratory worker, who was known to have positive reaction to coccidioidin skin testing, accidentally inoculated himself deep into a wrist bone with a suspension of "*C. immitis*" arthrospores. Coccidioidal osteomyelitis developed and persisted until the lesion was excised. Thereafter recovery proceeded without further complication. The sixth case was reported by Harrell and Honeycutt.[3] While visiting in Arizona, the patient pricked his thigh with a cactus thorn. Cutaneous coccidioidomycosis developed at the site. Data on all these cases and the one herein reported are given in Table 2.

What Wilson considered to be the nine basic criteria for making the diagnosis of cutaneous coccidioidomycosis have correlated well with subsequent cases reported in persons who never before had had a primary infection in the lungs or elsewhere. More recently Wilson[7] suggested the classification *cutaneous (chancriform) syndrome* for cases of primary skin infection with "*C. immitis*" or any other fungus capable of causing deep mycosis when inoculated into normal persons not previously infected by that organism.

The classification did not anticipate the further possibility of a person's becoming infected subcutaneously after previously having had a primary (and perhaps not clinically recognized) immunizing infection. Salkin,[4] however, in his "Pathogenetic Classification of Coccidioidomycosis," did consider the reinfection phase, submitting the term "exogenous reinfection" in contrast to "endogenous reinfection," as in the case of a "residual primary nodule" becoming an "abscessing coccidioma" years after the primary infection. Until Smith cited the case observed by Tiggert there had been no reference to reinfection in an immune subject.

There being no further information about that case, the case herein reported is of interest since we had opportunity to observe the clinical and pathological developments during the course of infection. It can be seen from Table 2 that it differed from the five cutaneous (chancriform) syndrome cases in several respects and paralleled the one case of exogenous reinfection in a number of pertinent features.

1. In our case the stimulation of the immune response was more rapid. Complement-fixing titer began to rise within the first week, while in those cases in which the test was done for the other groups it did not begin to rise before five weeks, and in some cases never. In our case the antibodies reached a peak in four weeks and began to recede. No precipitins developed that we could observe, yet in the other cases tested the reaction for precipitins was positive in three to five weeks.

2. The coccidioidin skin test was positive before reinfection in both our case and Tiggert's. There is no evidence of a preexisting skin reaction in any of the other group.

3. The regional lymphatic system at the infection site was not involved. There was no evidence of lymphangitis or lymphadenopathy, nor were there any other systemic developments except urticaria.

4. The infection site was well localized and was only 2 mm in diameter.

Each of these observed differences suggests that because of the previous primary (although subclinical) infection, the patient in the present case had acquired immunological resistance to reinfection and that there existed an immediate defensive power which prevented the development of the cutaneous (chancriform) syndrome. It seems logical to accept the classification suggested by Salkin and refer to such infections as cutaneous exogenous reinfections.

SUMMARY

This report presents the second known case of accidental cutaneous coccidioidal infection occurring in an immune person, the features of the case conforming to those of "exogenous reinfection."

The findings in previously reported cases of cutaneous inoculation coccidioidomycosis have been briefly summarized.

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